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NEWS EXPRESS JUNE 13 CURRENT WINDOWS VERSION IS V8.0, CURRENT MACINTOSH VERSION IS V6.0c(ENG) AND V6.0Jc(JP), AND CURRENT DISCOVER FILE IS DATED 13 JUNE 2005

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FILE LAST UPDATED: 16 Nov 2005 (20051116/ED)

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=> HEK293
L1 2736 HEK293

=> reovirus
1967 REOVIRUS
332 REOVIRUSES
L2 2036 REOVIRUS
(REOVIRUS OR REOVIRUSES)

=> reassorted
L3 49 REASSORTED

=> L2 and L3
L4 1 L2 AND L3

=> L1 and L4
L5 0 L1 AND L4

=> L1 and L2
L6 5 L1 AND L2

=> D L5 IBIB ABS 1-5
L5 HAS NO ANSWERS
L1 2736 SEA FILE=CAPLUS ABB=ON PLU=ON HEK293
L2 2036 SEA FILE=CAPLUS ABB=ON PLU=ON REOVIRUS
L3 49 SEA FILE=CAPLUS ABB=ON PLU=ON REASSORTED
L4 1 SEA FILE=CAPLUS ABB=ON PLU=ON L2 AND L3
L5 0 SEA FILE=CAPLUS ABB=ON PLU=ON L1 AND L4

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L6 ANSWER 1 OF 5 CAPLUS COPYRIGHT 2005 ACS on STN
 ACCESSION NUMBER: 2005:439832 CAPLUS
 DOCUMENT NUMBER: 143:227017
 TITLE: Inhibition of NF- κ B activity and cFLIP
 expression contribute to viral-induced apoptosis
 AUTHOR(S): Clarke, P.; DeBiasi, R. L.; Meintzer, S. M.; Robinson,
 B. A.; Tyler, K. L.
 CORPORATE SOURCE: Departments of Neurology, University of Colorado
 Health Sciences Center, Denver, CO, 80262, USA
 SOURCE: Apoptosis (2005), 10(3), 513-524
 CODEN: APOPFN; ISSN: 1360-8185
 PUBLISHER: Springer
 DOCUMENT TYPE: Journal
 LANGUAGE: English
 REFERENCE COUNT: 75 THERE ARE 75 CITED REFERENCES AVAILABLE FOR THIS
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=> D L6 IBIB abs 1-5

L6 ANSWER 1 OF 5 CAPLUS COPYRIGHT 2005 ACS on STN
 ACCESSION NUMBER: 2005:439832 CAPLUS
 DOCUMENT NUMBER: 143:227017
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AUTHOR(S): expression contribute to viral-induced apoptosis
 Clarke, P.; DeBiasi, R. L.; Meintzer, S. M.; Robinson,
 B. A.; Tyler, K. L.
 CORPORATE SOURCE: Departments of Neurology, University of Colorado
 Health Sciences Center, Denver, CO, 80262, USA
 SOURCE: Apoptosis (2005), 10(3), 513-524
 CODEN: APOPFN; ISSN: 1360-8185
 PUBLISHER: Springer
 DOCUMENT TYPE: Journal
 LANGUAGE: English

AB Virus-induced activation of nuclear factor-kappa B (NF- κ B) is required for Type 3 (T3) **reovirus**-induced apoptosis. We now show that NF- κ B is also activated by the prototypic Type 1 **reovirus** strain Lang (T1L), which induces significantly less apoptosis than T3 viruses, indicating that NF- κ B activation alone is not sufficient for apoptosis in **reovirus**-infected cells. A second phase of virus-induced NF- κ B regulation, where NF- κ B activation is inhibited at later times following infection with T3 Abney (T3A), is absent in T1L-infected cells. This suggests that inhibition of NF- κ B activation at later times post infection also contributes to **reovirus**-induced apoptosis. **Reovirus**-induced inhibition of stimulus-induced activation of NF- κ B is significantly associated with apoptosis following infection of **HEK293** cells with reassortant **reoviruses** and is determined by the T3 S1 gene segment, which is also the primary determinant of **reovirus**-induced apoptosis. Inhibition of stimulus-induced activation of NF- κ B also occurs following infection of primary cardiac myocytes with apoptotic (8B) but not non-apoptotic (T1L) **reoviruses**. Expression levels of the NF- κ B-regulated cellular FLICE inhibitory protein (cFLIP) reflect NF- κ B activation in **reovirus**-infected cells. Further, inhibition of NF- κ B activity and cFLIP expression promote T1L-induced apoptosis. These results demonstrate that inhibition of stimulus-induced activation of NF- κ B and the resulting decrease in cFLIP expression promote **reovirus**-induced apoptosis.

REFERENCE COUNT: 75 THERE ARE 75 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L6 ANSWER 2 OF 5 CAPLUS COPYRIGHT 2005 ACS on STN
 ACCESSION NUMBER: 2003:697089 CAPLUS
 DOCUMENT NUMBER: 139:207772
 TITLE: The use of ribozymes in the detection of adventitious agents for **reovirus** preparation useful in cancer therapy
 INVENTOR(S): Coffey, Matthew C.
 PATENT ASSIGNEE(S): Oncolytics Biotech Inc., Can.
 SOURCE: PCT Int. Appl., 26 pp.
 CODEN: PIXXD2
 DOCUMENT TYPE: Patent
 LANGUAGE: English
 FAMILY ACC. NUM. COUNT: 1
 PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2003072811	A2	20030904	WO 2003-CA264	20030226
WO 2003072811	A3	20040205		
W:	AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, OM, PH, PL, PT, RO, RU, SC, SD, SE, SG, SK, SL, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, YU, ZA, ZM, ZW			
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CA 2374388	AA	20030828	CA 2002-2374388	20020304
CA 2374388	C	20030828		
US 2004005546	A1	20040108	US 2003-375700	20030226

EP 1481084	A2	20041201	EP 2003-704136	20030226
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO, MK, CY, AL, TR, BG, CZ, EE, HU, SK				
BR 2003007882	A	20041228	BR 2003-7882	20030226
TR 200501513	T3	20050621	TR 2005-200501513	20030226
JP 2005518797	T2	20050630	JP 2003-571491	20030226
PRIORITY APPLN. INFO.:				
US 2002-360730P P 20020228				
US 2003-441760P P 20030123				
WO 2003-CA264 W 20030226				

AB The present invention provides a method of detecting adventitious agents in a composition comprising a microorganism by using ribozyme-expressing indicator cells, as well as indicator cells useful in such detection. The method is used to ensure that the **reovirus** preparation, used for tumor therapy, does not contain adventitious agents, which may result in undesired side effects. In particular, also disclosed is a method of preparing **reovirus** using mammalian cells (such as **HEK293** or **COS-1**) stably transfected with ribozyme, Rz-538 or Rz-984, which cleaves **reovirus** genome in case of the presence of adventitious agents.

L6 ANSWER 3 OF 5 CAPLUS COPYRIGHT 2005 ACS on STN

ACCESSION NUMBER: 2003:366194 CAPLUS

DOCUMENT NUMBER: 139:20428

TITLE: Two Distinct Phases of Virus-induced Nuclear Factor κ B Regulation Enhance Tumor Necrosis Factor-related Apoptosis-inducing Ligand-mediated Apoptosis in Virus-infected Cells

AUTHOR(S): Clarke, Penny; Meintzer, Suzanne M.; Moffitt, Lisa A.; Tyler, Kenneth L.

CORPORATE SOURCE: Departments of Neurology, University of Colorado Health Science Center, Denver, CO, 80220, USA

SOURCE: Journal of Biological Chemistry (2003), 278(20), 18092-18100

PUBLISHER: American Society for Biochemistry and Molecular Biology

DOCUMENT TYPE: Journal

LANGUAGE: English

AB Cellular transcription factors are often utilized by infecting viruses to promote viral growth and influence cell fate. The authors have previously shown that nuclear factor κ B (NF- κ B) is activated after **reovirus** infection and that this activation is required for virus-induced apoptosis. In this report the authors identify a second phase of **reovirus**-induced NF- κ B regulation. The authors show that at later times post-infection NF- κ B activation is blocked in **reovirus**-infected cells. This results in the termination of virus-induced NF- κ B activity and the inhibition of tumor necrosis factor α and etoposide-induced NF- κ B activation in infected cells. **Reovirus**-induced inhibition of NF- κ B activation occurs by a mechanism that prevents I κ B α degradation and that is blocked in the presence of the viral RNA synthesis inhibitor, ribavirin. **Reovirus**-induced apoptosis is mediated by tumor necrosis factor-related apoptosis inducing ligand (TRAIL) in a variety of epithelial cell lines. Herein the authors show that ribavirin inhibits **reovirus**-induced apoptosis in TRAIL-resistant **HEK293** cells and prevents the ability of **reovirus** infection to sensitize TRAIL-resistant cells to TRAIL-induced apoptosis. Furthermore, TRAIL-induced apoptosis is enhanced in **HEK293** cells expressing I κ B α N2, which blocks NF- κ B activation. These results indicate that the ability of **reovirus** to inhibit NF- κ B activation sensitizes **HEK293** cells to TRAIL and facilitates virus-induced apoptosis in TRAIL-resistant cells. These findings demonstrate that two distinct phases of virus-induced NF- κ B regulation are required to efficiently activate host cell apoptotic responses to **reovirus** infection.

REFERENCE COUNT: 62 THERE ARE 62 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L6 ANSWER 4 OF 5 CAPLUS COPYRIGHT 2005 ACS on STN

ACCESSION NUMBER: 2002:607491 CAPLUS
DOCUMENT NUMBER: 138:54436
TITLE: **Reovirus**-induced apoptosis requires both
death receptor- and mitochondrial-mediated
caspase-dependent pathways of cell death
Kominsky, D. J.; Bickel, R. J.; Tyler, K. L.
Corporate Source: Department of Neurology, University of Colorado Health
Science Center, Denver, CO, 80262, USA
Source: Cell Death and Differentiation (2002), 9(9), 926-933
CODEN: CDDIEK; ISSN: 1350-9047

PUBLISHER: Nature Publishing Group
DOCUMENT TYPE: Journal
LANGUAGE: English

AB Apoptosis plays an important role in the pathogenesis of many viral infections. Despite this fact, the apoptotic pathways triggered during viral infections are incompletely understood. The authors now provide the first detailed characterization of the pattern of caspase activation following infection with a cytoplasmically replicating RNA virus.

Reovirus infection of **HEK293** cells results in the activation of caspase-8 followed by cleavage of the pro-apoptotic protein Bid. This initiates the activation of the mitochondrial apoptotic pathway leading to release of cytochrome c and activation of caspase-9. Combined activation of death receptor and mitochondrial pathways results in downstream activation of effector caspases including caspase-3 and caspase-7 and cleavage of cellular substrates including PARP. Apoptosis is initiated by death receptor pathways but requires mitochondrial amplification producing a biphasic pattern of caspase-8, Bid, and caspase-3 activation.

REFERENCE COUNT: 40 THERE ARE 40 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L6 ANSWER 5 OF 5 CAPLUS COPYRIGHT 2005 ACS on STN

ACCESSION NUMBER: 2002:209874 CAPLUS
DOCUMENT NUMBER: 136:382804
TITLE: **Reovirus**-induced alterations in gene
expression related to cell cycle regulation
Poggioli, George J.; DeBiasi, Roberta L.; Bickel,
Ryan; Jotte, Robert; Spalding, Aaron; Johnson, Gary
L.; Tyler, Kenneth L.

CORPORATE SOURCE: Department of Microbiology, University of Colorado
Health Sciences Center, Denver, CO, 80220, USA

SOURCE: Journal of Virology (2002), 76(6), 2585-2594
CODEN: JOVIAM; ISSN: 0022-538X

PUBLISHER: American Society for Microbiology
DOCUMENT TYPE: Journal
LANGUAGE: English

AB Mammalian **reovirus** infection results in perturbation of host cell cycle progression. Since **reovirus** infection is known to activate cellular transcription factors, we investigated alterations in cell cycle-related gene expression following **HEK293** cell infection by using the Affymetrix U95A microarray. Serotype 3 **reovirus** infection results in differential expression of 10 genes classified as encoding proteins that function at the G1-to-S transition, 11 genes classified as encoding proteins that function at G2-to-M transition, and 4 genes classified as encoding proteins that function at the mitotic spindle checkpoint. Serotype 1 **reovirus** infection results in differential expression of four genes classified as encoding proteins that function at the G1-to-S transition and three genes classified as encoding proteins that function at G2-to-M transition but does not alter any genes classified as encoding proteins that function at the mitotic spindle checkpoint. We have previously shown that serotype 3, but not serotype 1, **reovirus** infection induces a G2-to-M transition arrest resulting from an inhibition of cdc2 kinase activity. Of the differentially expressed genes encoding proteins regulating the G2-to-M transition, *chk1*, *weel*, and *GADD45* are known to inhibit cdc2 kinase activity. A hypothetical model describing serotype 3 **reovirus**-induced inhibition of cdc2 kinase is presented, and **reovirus**-induced perturbations of the G1-to-S, G2-to-M, and mitotic spindle checkpoints are discussed.

REFERENCE COUNT:

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<u>L11</u>	L9 and L5L10	0	<u>L11</u>
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<u>L7</u>	L6 and reassorted	0	<u>L7</u>
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<u>L5</u>	Coffey M.in.	69	<u>L5</u>
<u>L4</u>	L3 and HEK293	0	<u>L4</u>
<u>L3</u>	reassorted adj reovirus	5	<u>L3</u>
<u>L2</u>	assorted adj reovirus	0	<u>L2</u>
<u>L1</u>	assorted adj revirus	0	<u>L1</u>

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